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Modern Concepts of Cardiovascular Disease

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DIET AND HEART FAILURE

Diet is given a comparatively small role in the therapeutic armamentarium of heart failure. This is probably because so little has really been known of the effects of various diets in heart disease. A study of the literature over the past twenty years, both in this country and abroad, discloses an abundance of notions regarding diet in heart disease, but strikingly little clinical evidence to support them. By and large, dietary recommendations have been based either upon empiric observations, simple impressions, or the dicta of previous authorities.

In the more severe stage of congestive heart failure, the Karell diet enjoys a widespread use. As improvement sets in, little attention is customarily paid to food, other than to manipulate the quantity according to the desires and comfort of the patient. In general, however, it seems agreed that in the presence of myocardial weakness the intake of both fluids and food should be restricted. Such restrictions, however, have as a rule been guided more by the empiricism of symptomatic relief than by a specific knowledge of the effect of the food or fluid restriction upon the metabolic and circulatory state in heart disease.

That food restriction has striking circulatory and metabolic effects in normal people has been known for some twenty years, since the classic studies of Benedict, who found that undernutrition over the course of several weeks produced among other things a slowing of the pulse rate, a fall in both systolic and diastolic blood pressures, a drop in the basal metabolic rate, and a drop in the level of fluid exchange as measured by fluid intake and urinary output. All this was obtained after a loss of about 10% of the normal body weight, without significant impairment of vigor and without the necessity for restriction of activities. A striking example of what food restriction alone will do may be observed in cases of so-called anorexia nervosa, in which the whole level of life, as measured by the circulatory state and metabolic and fluid exchanges, is greatly depressed, often without disturbing the patient's normal daily activities.

It is evident that such circulatory and metabolic changes which can be induced by dietary restriction are highly desirable in patients with myocardial weakness. Thus, slowing of the heart rate permits the usually thickened heart muscle a greater opportunity for recovery from muscular contraction during the period of the prolonged diastole; a decrease in the level of oxygen consumption lessens the need for the heart to supply oxygen to the tissues; a lowering of the blood pressure further lessens the work of the heart by lowering the resistance against which it must propel the blood; and finally, the low level of fluid exchange may act favorably by diminishing the amount of blood to be forced around the vascular circuit.

That such predictable benefits actually occur has been demonstrated experimentally in heart failure from various causes (rheumatic endocarditis, luetic aortitis with aortic regurgitation, arteriosclerotic heart disease, and concretio cordis). The results of these experiments on food restriction in patients with heart failure have been described elsewhere by H. Magendantz and the author. In this study, which dealt with patients who had heart failure, it was extremely difficult to evaluate results from experimental procedures, largely because heart failure is a dynamic state and, as such, subject to considerable spontaneous and often uncontrollable fluctuations in one direction or the other. In general the procedure which we employed was first to enforce absolute bed rest with sedatives, usually morphia, until the state of the circulation seemed fairly stationary, then to administer digitalis, all other conditions remaining fixed. When the full digitalis effect had been obtained and another stationary state occurred, the dietary restriction was begun, first severe, then gradually more moderate, so that the patients lost about 10% of their body weight in from three to four weeks. Thereafter, they were placed on a maintenance diet. During the period of undernutrition, fluids were unrestricted; however, the patients spontaneously drank less.

The effect of this program of undernutrition on the cardiorespiratory state was marked in most

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JAN 2 6

cases, but was not parallel to the degree of weight loss. The heart rate became considerably slower. This slowing occurred whether there was initially a normal sinus rhythm or auricular fibrillation. The degree of the slowing as the result of the diet restriction was beyond what could be obtained with digitalis and bed rest alone. Also, there was a significant decrease in the systolic and diastolic blood pressures. This, together with a decrease in the cardiac output indicated a great lessening in the level of cardiac work. Another definite index of the gain in strength of the heart muscle was reflected in the increase in vital capacity which could be directly attributable to the weight reduction. This was accompanied by a decrease in pulmonary ventilation as well. In some patients lowering of the venous pressure likewise was noted. Probably the most significant change indicating improvement under these conditions of food restriction was the measurable decrease in the size of the heart.

Some of the patients whom we studied were observed during periods of graded exercise before and after dietary restriction. From a study of pulse and respiratory rate, pulmonary ventilation, and oxygen consumption, it was apparent that the response of these patients to exercise showed changes indicative of improvement, and corresponding to those obtained during rest.

The velocity of blood flow as measured by arm-to-tongue time was not affected. The electrocardiograms remained unchanged during the period of study. The metabolic exchanges of water, nitrogen, and sodium chloride were observed during the experimental period. Naturally a negative nitrogen balance prevailed, but this was in no way proportional to the improvement noted in the circulatory state. There was a decreased output of urinary sodium chloride, obviously due to the decreased intake in the diet. Occasionally there was a decrease in the level of fluid exchange, that is, the intake of fluid and output of urine, although usually this level was not strikingly altered.

In one case during prolonged undernutrition the globulin rose steadily; this was followed by a late fall in the serum albumin concentration, producing an actual reversal of the albumin-globulin ratio. In spite of the low calcium content of the diet, the calcium concentration of the serum remained unchanged. Similarly, although the iron content of the experimental diet was low, the red blood cells and hemoglobin concentration were stationary. (There may have been an absolute decreased masked by a simultaneous fall in plasma volume.) No albuminuria was noted.

As for the subjective effect of the restricted diet, hunger and weakness were most prominent. The former complaint was effectively combatted by sedation at first, then as improvement occurred, with suggestion and explanation of the purpose of the underfeeding. In general the loss of nitrogen appeared to parallel the intensity of the hunger. While there were occasional subjective complaints of weakness, it is interesting that muscular strength as measured with a dynamometer showed no alteration even after prolonged undernutrition. Subjective weakness, which may result from undernutrition, might conceivably be beneficial in automatically restricting physical activities. When the maintenance diet was given, weakness regularly disappeared.

The measurable improvement in the cardiovascular status continued only so long as the lower weight level was maintained. As weight was regained, the degree of improvement diminished.

Favorable results are not regularly obtained with a restricted diet. This is particularly true in patients in congestive failure who have already lost considerable weight. It appears also that in obese patients, while reduction is unquestionably desirable, the physiological effect of undernutrition is not as great as in people of normal size. Patients of normal size also vary considerably in their response to weight reduction. This was indicated in Benedict's work and is simply further evidence of the fact that human beings rarely respond uniformly to any experimental procedures.

As indicated above, there is a tendency, even in those who have experienced striking benefit from food restriction, for the beneficial effect to disappear even with a partial regaining of weight. Because of this, however, it does not follow that the food restriction is of no value. It would be just as illogical to conclude that rest in bed is of no value to a patient with heart failure because eventually he must be out of bed. During the stage of congestive heart failure, any measure which will reduce the work of the heart is desirable, and food restriction is definitely such a measure. When heart failure has been overcome and circulatory balance reëstablished, extreme restrictive measures are no longer necessary.

That food restriction has certain specific beneficial effects in stages of heart disease other than heart failure has been demonstrated by the work of Master, Jaffe and Dack, who have presented evidence to indicate that in patients with coronary thrombosis dietary restriction may be very helpful.

A discussion of food regulation in patients with heart disease naturally leads to a consideration of the role of water and salt. In this connection may be mentioned recent observations which Magendantz, Ginsburg, and the author have made. We have found that during various stages of heart failure, restriction of water alone seems unnecessary, inasmuch as the amount of water, as such, which the patient can comfortably take seems to have no harmful effect on the circulatory state in the presence of cardiac weakness. Sodium chloride, however, even in slightly excessive amounts, seems to have a strikingly aggravating effect, as little as 15 grams a day often producing in three or four days a picture of severe congestive failure where only mild symptoms of failure had previously been present. This would indicate that as regards water and salt, comparatively little attention need be paid to regulating the fluid intake in patients with cardiac weakness, if care is taken to keep the salt intake low.

In summary, then, it may be said that rigid dietary restriction produces unquestioned beneficial effects on the state of the circulation in patients with heart failure and that these effects are of about the same magnitude and in the same direction as those obtained following digitalis. The dietary restriction should be such as to effect a loss of about 10% of body weight (exclusive of edema fluid) over a period of two to four weeks. The inconveniences or possible harmful effects of this dietary regime are negligible and the beneficial effects will persist so long as the lower weight level is maintained. It appears that the beneficial effects on the circulation are even more striking when weight loss occurs from a normal level than when the reduction is from an obese level. While no special attention is required in the regulation of water intake as such, it is important to avoid excessive salt intake.

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